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TECHNICAL PROGRESS REPORT

DEFENSE ATOMIC SUPPORT AGENCY
WASHINGTON 25, D. C.

(5) 577 .500

(6) TERTIARY BLAST EFFECTS:
THE EFFECTS OF IMPACT ON MICE, RATS,
GUINEA PIGS AND RABBITS.

(10) by

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(9) Technical Progress Report,
on
(15) Contract ~~No.~~ DA-49-146-XZ-055

This work, an aspect of investigations dealing with the Biological Effects of Blast from Bombs, was supported by the Defense Atomic Support Agency of the Department of Defense.

Lovelace Foundation for Medical Education and Research
Albuquerque, New Mexico

February 28, 1961

TERTIARY BLAST EFFECTS:
THE EFFECTS OF IMPACT ON MICE, RATS,
GUINEA PIGS AND RABBITS

FORWORD

The present report, though related to blast and shock biology, deals with the results of exposure of four species of animals to impact. Extrapolation of the mortality data to the 70 kg animal and a comparison of the results with relevant information in the literature dealing with human response to dynamic accelerative or decelerative loading is presented.

The results are limited to situations in which impact with a hard surface occurs and therefore to circumstances wherein only the animals own tissues are active in absorbing the energy of motion, i.e., the time and distance over which energy dissipation occurs is minimal, a fact which tends to maximize the impact load. These findings are applicable to many situations in which injury may occur either from the impact of blunt objects striking a biological target or from a moving target striking a solid object.

The impact study represents a segment of experimentation which has been under way since 1952 aimed at clarifying the biological response following exposure to blast phenomena including overpressures, winds, moving debris, and ground shock.

ABSTRACT

A total of 455 mice, rats, guinea pigs and rabbits were subjected to impact at velocities ranging between 25 ft/sec and 51 ft/sec. The desired velocities were generated by allowing the animals to free-fall from various heights to a flat concrete pad. The ventral surface of each animal was the area of impact.

Probit analyses of the 24-hr mortality data yielded LD_{50} impact velocities with 95 per cent confidence limits as follows: mouse, 39.4 (37.4 - 42.0) ft/sec; rat, 43.5 (42.0 - 44.8) ft/sec; guinea pig, 31.0 (30.0 - 31.9) ft/sec; and rabbit, 31.7 (30.2 - 33.3) ft/sec. The LD_{50} figures for the mouse and rat were significantly higher, statistically, than those for the guinea pig and rabbit.

The small spread in the LD_{50} values suggested little variation in the tolerance of biological systems to impact. Further, the steepness of the mortality curves indicated a narrow survival range to impact.

Extrapolation of the experimental data to the 70 kg animal yielded a predicted LD_{50} impact velocity of 26 ft/sec (18 mph). Literature relevant to the human case was reviewed and the tentative applicability of the predicted figures to adult man was discussed.

ACKNOWLEDGEMENTS

The authors are indebted to the following individuals for their participation in this project: Miss Faith Sherping, V. C. Goldizen, D.E.Pratt, V.R.Clare, R.W.Albright, C.C.Fisher, R.T.Sanchez, A.F.Strehler, R.V.Taborelli, M.B.Wetherbe and J.A.Henry. Appreciation is also expressed to R.A.Smith, G.S.Bevil and Mrs. Holly Ferguson for the preparation of the illustrative material, and to Mrs. Isabell Benton and Mrs. Beth Brown for editorial and secretarial aid.

This work was initially supported by contract with the Division of Biology and Medicine of the Atomic Energy Commission and finally by contract with the Defense Atomic Support Agency of the Department of Defense.

SUMMARY

1. A total of 455 animals including 113 mice, 178 rats, 111 guinea pigs and 53 rabbits were subjected to impact at velocities ranging between 25 ft/sec and 51 ft/sec.

2. The desired impact velocities were generated by allowing the animals to free-fall from various heights to a flat concrete pad. The ventral surface of each animal was the area of impact.

3. The velocities at impact were determined from equations that were empirically derived from high speed photographic records of the animals at impact.

4. Probit analyses of the 24-hr mortality data yielded LD_{50} values with 95 per cent confidence limits as follows: mouse, 39.4 ft/sec (37.4 - 42.0); rat, 43.5 ft/sec (42.0 - 44.8); guinea pig, 31.0 ft/sec (30.0 - 31.9); and rabbit, 31.7 ft/sec (30.2 - 33.3).

5. Of the 200 animals killed by impact, 149 (75 per cent) died within 20 min and 90 per cent within one hour. Only 10 per cent of the deaths occurred between the 2-hr and 24-hr period. The general trend was for the larger species to have the longer survival times.

6. From an interspecies extrapolation the LD_{50} impact velocity for a 70 kg animal was calculated to be 26 ft/sec (18 mph).

7. A probit mortality curve was calculated for a 70 kg animal to predict threshold conditions for lethality which was 21 ft/sec (14 mph).

8. The results from the present study were discussed revelant to the information available in the literature on the effects of ground shock on personnel in underground structures, deck heave, translation caused by air blast, automobile accidents, falls, and related decelerative phenomena.

9. The minimum impact velocity required for skull fracture was pointed out to be near 13.5 ft/sec (9.2 mph). (Gurdjian et al.)

10. The "initial velocity" threshold for fracture of the heel bone of standing objects was between 11 and 16 ft/sec (Black et al.; Draeger et al.).

11. The maximal impact velocity tolerated by human subjects, dropped in a seated position, was reported to be about 10 ft/sec (Swearingen et al.).

12. Human fatalities in automobile statistics showed 50 per cent mortality at vehicular speeds near 33.8 ft/sec (23 mph) which was in fair agreement with the 50 per cent impact velocity (26 ft/sec) obtained in the present study for an animal of comparable body weight (from DeHaven).

13. It was tentatively concluded that 10 ft/sec (7 mph) was the "on-the-average safe" impact for adult humans.

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INTRODUCTION

To serve the purposes of study and presentation, the biological effects of air blast have been arbitrarily divided into several categories, the most important of which are primary, secondary, and tertiary effects.[1-4] Primary damage is that associated with variations in environmental pressure per se. Injuries generally occur where the variation in tissue density is the greatest, and in particular, involve the air-containing organs; e.g., the sinuses, ears, lungs, and gastrointestinal tract. When the lungs are significantly injured, widespread arterial air emboli ensue and frequently produce rapid mortality when blood flow in coronary and cerebral vessels is embarrassed.[1-8]

Secondary effects include those injuries resulting from the impact of penetrating or nonpenetrating missiles energized by blast pressures, winds, ground shock, and gravity. A wide variety of injuries is seen ranging from slight lacerations to penetrating and perforating lesions due to flying debris, including fragments of glass and other frangible materials. Also, massive, crushing injuries can occur from the collapse of inhabited structures of various types.

Tertiary effects encompass injuries that occur as a consequence of actual displacement of a biological target by winds that accompany the propagation of the pressure pulse. Though damage may ensue during the accelerative phase of movement because of differential velocities imparted to various portions of the body, trauma is likely to be more prevalent and severe during deceleration, particularly if impact with a hard surface occurs. Injuries in this category may be somewhat similar to those mentioned above for secondary effects and may frequently bear a resemblance to those observed in victims of automobile accidents,[9,10] falls,[11] and airplane crashes;[12,13] e.g., abrasions, lacerations, contusions, fractures, and rupture of, and damage to, the internal organs, including the heart, lungs, liver, spleen, brain, and spinal cord.

Proper assessment of the tertiary blast hazard requires knowledge in at least two areas; namely, (a) information concerning velocities attained by objects the size and shape of man in relation to the physical parameters of the blast

wave, and (b) man's tolerance to impact as a function of striking velocity. The former has been studied by Taborelli et al.[14] in full-scale nuclear tests, and Bowen and co-workers[15] have formulated a mathematical model for predicting the velocity-history of objects as large as man when energized by blast pressures and winds from modern high-yield explosions. Relatively little, however, is known quantitatively about the biology of decelerative impact referable either to humans or other mammals under circumstances wherein the stopping time and distance — other things being equal — are primary functions of the organism itself and not modified by other factors, such as deformation of vehicular structures, indentations in "soft" surfaces, and other events serving to depress the peak G load that develops during deceleration.

Because of this fact a relevant exploratory investigation using experimental animals was planned, carried out and the data assessed as one possible means of gaining some quantitative insight into the tolerance of man to impact. The following material will first describe the experiments performed; second, detail the observed "dose"-response relationship between velocity at impact and lethality for mice, rats, guinea pigs, and rabbits striking a flat concrete surface in the ventral position; third, set forth an interspecies comparison noting the association between average body weight and impact velocity responsible for mortality in each species; and last, briefly discuss the implications of the data with regard to extrapolation to the human case.

METHODS

1. Generation of Impact Velocities

The necessary range of velocities was obtained by dropping animals from different heights onto a flat concrete slab. Animals were released, one at a time, from a small box hoisted by a cable-pulley system attached to a 54 ft pole. The bottom of the box was opened by means of a solenoid-operated mechanism. At lower heights some of the animals were released by hand. Animals were in the prone position when dropped and when they struck the concrete pad. The height of drop was measured from the ventral surface of the animal's trunk to the surface of the impact area.

2. Animals

In all, a total of 455 animals were dropped in this study; their mean body weights, standard deviation, and the weight ranges are given in Table 1. There were 113 mice and 178 rats dropped at intervals between 15 ft and 54 ft; 111 guinea pigs from heights between 10 ft and 24 ft; and 53 rabbits between 12 ft and 28 ft.

The animals killed by impact were autopsied* as soon after death as possible, while survivors were sacrificed and autopsied after 24 hrs. The mortality figures reported subsequently, therefore, represent lethality up to 24 hrs.

3. Determination of Impact Velocities

Initially, impact velocities were determined from the timing marks on a Fastax camera film record taken of the animals just before impact. Velocities so determined for animals dropped from several different heights showed that the four species did not attain the same velocity for a given height of fall. Since it was impractical to take motion pictures of all the animals at impact, it was necessary to derive equations that would allow the calculation of the impact velocities.

Details of the experimental procedure and the derivation of the equations are reported elsewhere.[16] Briefly, the procedure was as follows:

An acceleration coefficient, alpha (α), was experimentally determined for freely falling objects including the four species of animals concerned here. Alpha was defined as the area presented to the wind stream times the object's drag coefficient divided by its mass. The following empirical relation between alpha and mass was obtained for small animal species:

$$\log \alpha = 0.01153 - 0.32400 \log m \qquad (1)$$

where

α = acceleration coefficient in ft^2/lb

m = animal's mass in grams

*The gross pathology observed in the animals subjected to impact will be the subject of a separate report.

Table I

ANIMALS USED IN THIS STUDY

Species	Number	Mean body weight	Range	Standard deviation
Mice	113	19.8 g	(16-28)	3.8 g
Rats	178	185 g	(150-250)	29 g
Guinea pigs	111	650 g	(480-811)	162 g
Rabbits	53	2.43 kg	(1.62-3.63)	0.47 kg
Total	455			

The following relationship for impact velocity was also experimentally derived:

$$V = (2gH)^{1/2} \left[(1 - e^{-\rho\alpha H})/\rho\alpha H\right]^{-1/2} \qquad (2)$$

where

V = impact velocity
g = acceleration of gravity
H = height of fall
ρ = air density
α = acceleration coefficient

Thus, the alpha for each group of animals dropped at the different heights, as reported in Tables 2 through 5, was calculated by substituting the appropriate mean mass (body weight) into equation (1). Solving equation (2) with the proper values of α, g, H, and ρ yielded impact velocities for each group. The values so obtained for impact velocities were carefully checked in individual animals for each species and were consistent with the data obtained using high speed photography.

RESULTS

1. Mortality

The 24-hr mortality data observed for mice, rats, guinea pigs, and rabbits are presented in Tables 2, 3, 4 and 5, respectively. Each table gives the mortality associated with the height of the fall and the computed impact velocity over the range in lethality from near zero to about 100 per cent for each species. Thus, the empirical data establishes a "dose"-response relationship for each species of animal.

To further assess this relationship an appropriate program for a Bendix G-15 Computer was prepared to apply the probit analysis of Finney[17] to the data presented in Tables 2 through 5. The probit transformation relates the percent mortality in probit units to the log of the "dose" – the "dose" here being the velocity at impact–and allows a sigmoid response curve to be expressed as a linear regression equation of the general form:

Table 2

THE RELATION BETWEEN
MOUSE MORTALITY AND IMPACT VELOCITY

Height of drop ft	Impact velocity ft/sec	Number dead over the number dropped	Mortality %
15	28.4	0/10	0
18	30.8	1/10	10
21	32.6	3/10	30
28	36.3	6/20	30
32	38.5	6/22	27
36	39.3	3/11	27
42	41.3	7/10	70
48	43.0	8/10	80
54	45.3	10/10	100
	Total	44/113	

computed LD_{50} = 39.4 ft/sec

Table 3

THE RELATION BETWEEN
RAT MORTALITY AND IMPACT VELOCITY

Height of drop ft	Impact velocity ft/sec	Number dead over the number dropped	Mortality %
15	29.8	0/10	0
18	32.3	0/10	0
21	34.6	0/10	0
24	36.8	1/10	10
27	38.7	2/10	20
30	40.4	3/10	30
33	42.0	6/10	60
36	43.6	2/10	20
39	45.3	9/20	45
42	46.5	23/26	88
45	47.5	8/10	80
48	48.6	8/10	80
51	49.8	8/10	80
54	50.9	20/22	91
	Total	90/178	

computed LD_{50} = 43.5 ft/sec

Table 4

THE RELATION BETWEEN
GUINEA PIG MORTALITY AND IMPACT VELOCITY

Height of drop ft	Impact velocity ft/sec	Number dead over the number dropped	Mortality %
10	24.8	0/10	0
12	27.0	2/10	20
13	28.1	0/4	0
14	29.1	1/10	10
15	30.0	4/10	40
16	30.9	6/12	50
17	31.9	5/10	50
18	32.7	8/10	80
19	33.6	8/11	73
20	34.4	9/10	90
21	35.1	10/10	100
24	37.2	4/4	100
	Total	57/111	

computed LD_{50} = 31.0 ft/sec

Table 5

THE RELATION BETWEEN
RABBIT MORTALITY AND IMPACT VELOCITY

Height of drop ft	Impact velocity ft/sec	Number dead over the number dropped	Mortality %
12	27.4	0/10	0
14	29.5	2/10	20
16	31.5	5/10	50
18	33.3	7/10	70
20	35.1	9/10	90
22	36.7	1/1	100
24	38.2	1/1	100
28	41.2	1/1	100
	Total	26/53	
	computed LD_{50} = 31.7 ft/sec		

$$Y = a + b \log X$$

where

Y = percent mortality in probit units

X = velocity of impact in ft/sec

a = constant for the intercept

b = slope constant for the regression line

The results of the probit analyses are presented graphically for each species in Figs. 1 through 4. Each figure notes the regression equation appropriate to the species of animal and shows the regression line, the grouped individual data points, the 95 per cent confidence limits of the information and the LD_{50} "velocity-dose" figure in ft/sec which is that impact velocity associated with 50 per cent mortality obtained by substituting 5 (the probit unit equal to 50 per cent mortality) for Y and solving the regression equation for X.

Similarly, impact velocity values associated statistically with any percent mortality may be calculated, as was done, for example, for 10 and 90 per cent mortality as noted in Table 6 comparing the results for the four species of animals employed. The table also presents the values for the regression equation intercepts and slope constants, the standard error of the slope constant and the 95 per cent confidence limits of the impact-velocity figures.

The solid lines in Fig. 5 set forth a graphic comparison of data noted in Table 6. As far as the impact velocity figures associated with 50 per cent mortality are concerned, it can be said that the LD_{50} value of 31.0 ft/sec for the guinea pig was not significantly different from that for the rabbit of 31.7 ft/sec. Those for the mouse (39.4 ft/sec) and rat (43.5 ft/sec), however, were statistically different from one another at the 95 per cent confidence limit; likewise, the LD_{50}'s for the guinea pig and rabbit were significantly below those for either the mouse or the rat at the 95 per cent confidence limit.

Concerning the variability in the slope constants, it may be stated

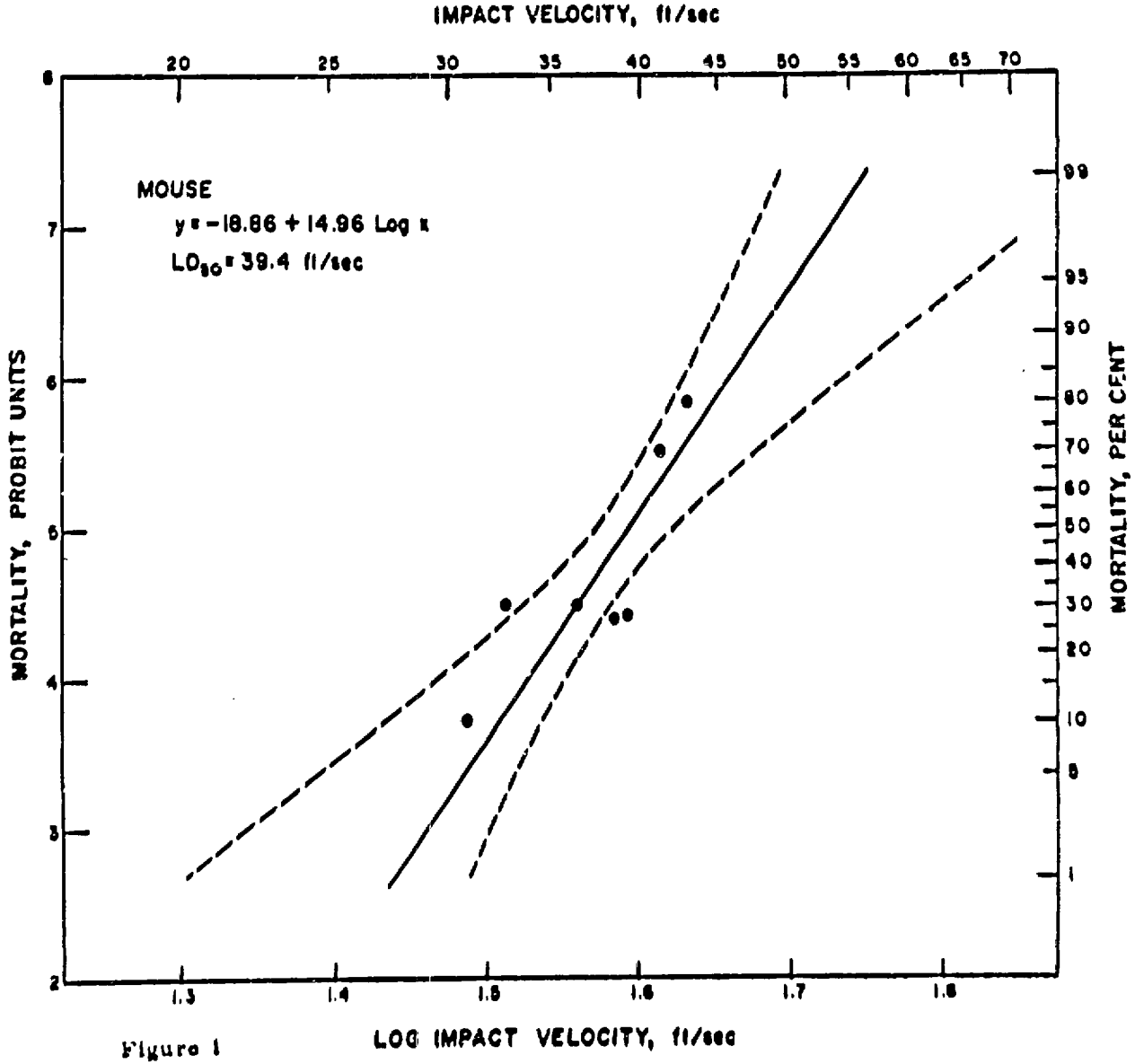
IMPACT VELOCITY, ft/sec
20
25
30
35
40
45
50
55
60
65
70
MOUSE
y = -18.86 + 14.96 Log x
LD50 = 39.4 ft/sec
MORTALITY, PROBIT UNITS
MORTALITY, PER CENT
99
95
90
80
70
60
50
40
30
20
10
5
1
2
3
4
5
6
7
8
1.3
1.4
1.5
1.6
1.7
1.8
LOG IMPACT VELOCITY, ft/sec

Figure 1

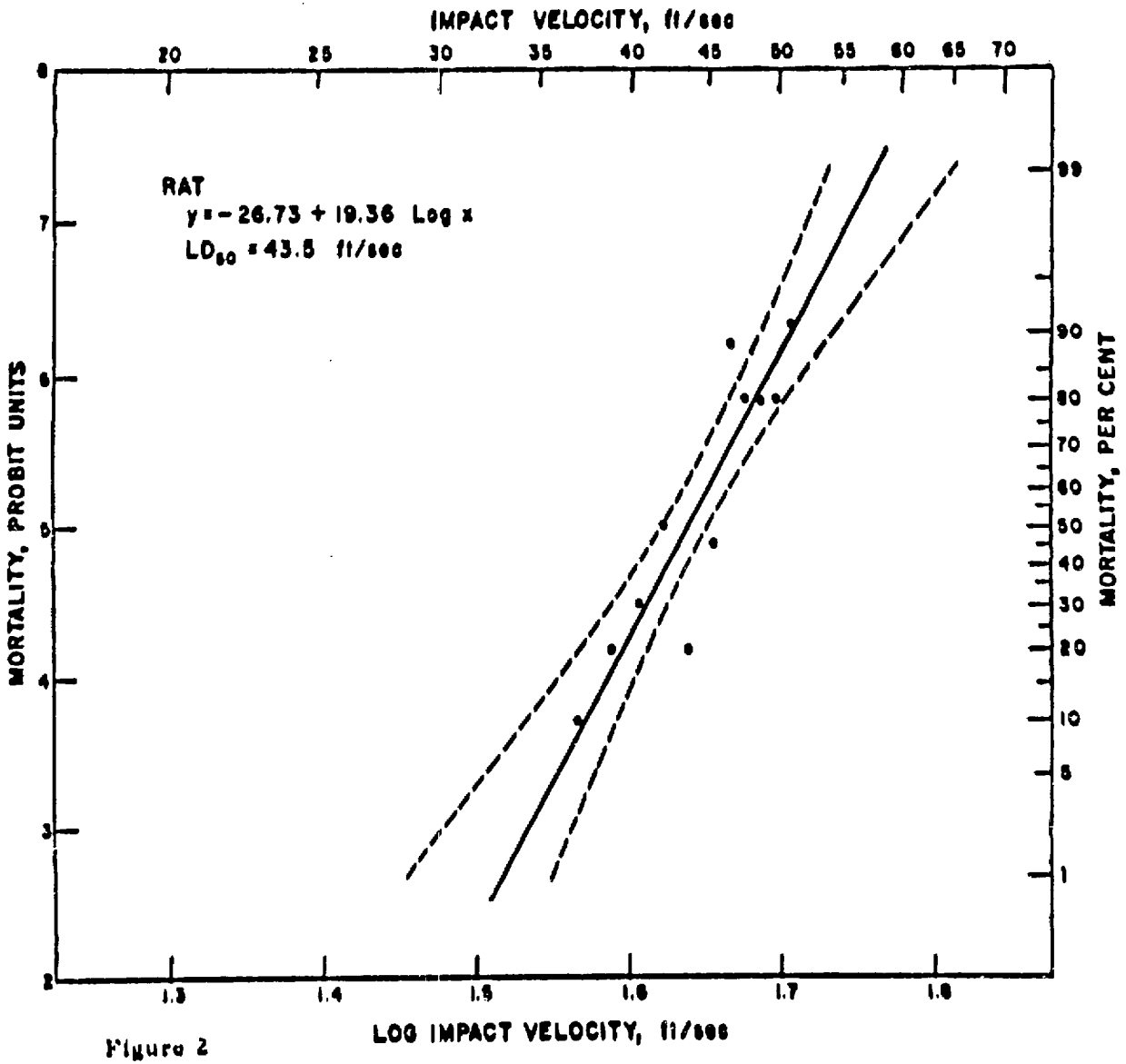
IMPACT VELOCITY, ft/sec
20
25
30
35
40
45
50
55
60
65
70
RAT
y = - 26.73 + 19.36 Log x
LD50 = 43.5 ft/sec
MORTALITY, PROBIT UNITS
MORTALITY, PER CENT
99
90
80
70
60
50
40
30
20
10
5
1
8
7
6
5
4
3
2
1.3
1.4
1.5
1.6
1.7
1.8
LOG IMPACT VELOCITY, ft/sec

Figure 2

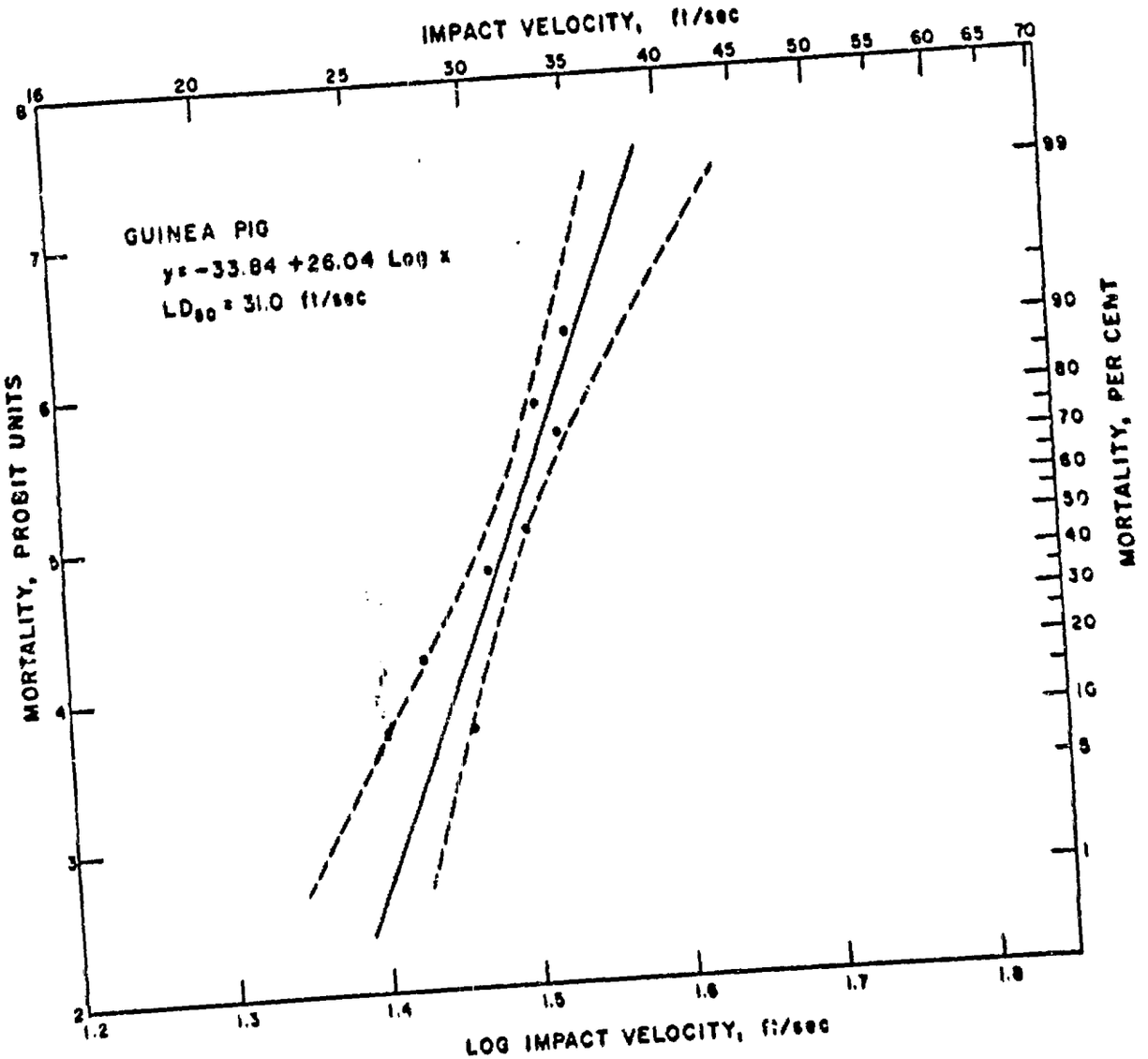
IMPACT VELOCITY, ft/sec
20
25
30
35
40
45
50
55
60
65
70
GUINEA PIG
y = -33.84 +26.04 Log x
LD50 = 31.0 ft/sec
MORTALITY, PROBIT UNITS
MORTALITY, PER CENT
99
90
80
70
60
50
40
30
20
10
5
1
1.2
1.3
1.4
1.5
1.6
1.7
1.8
LOG IMPACT VELOCITY, ft/sec

Figure 3

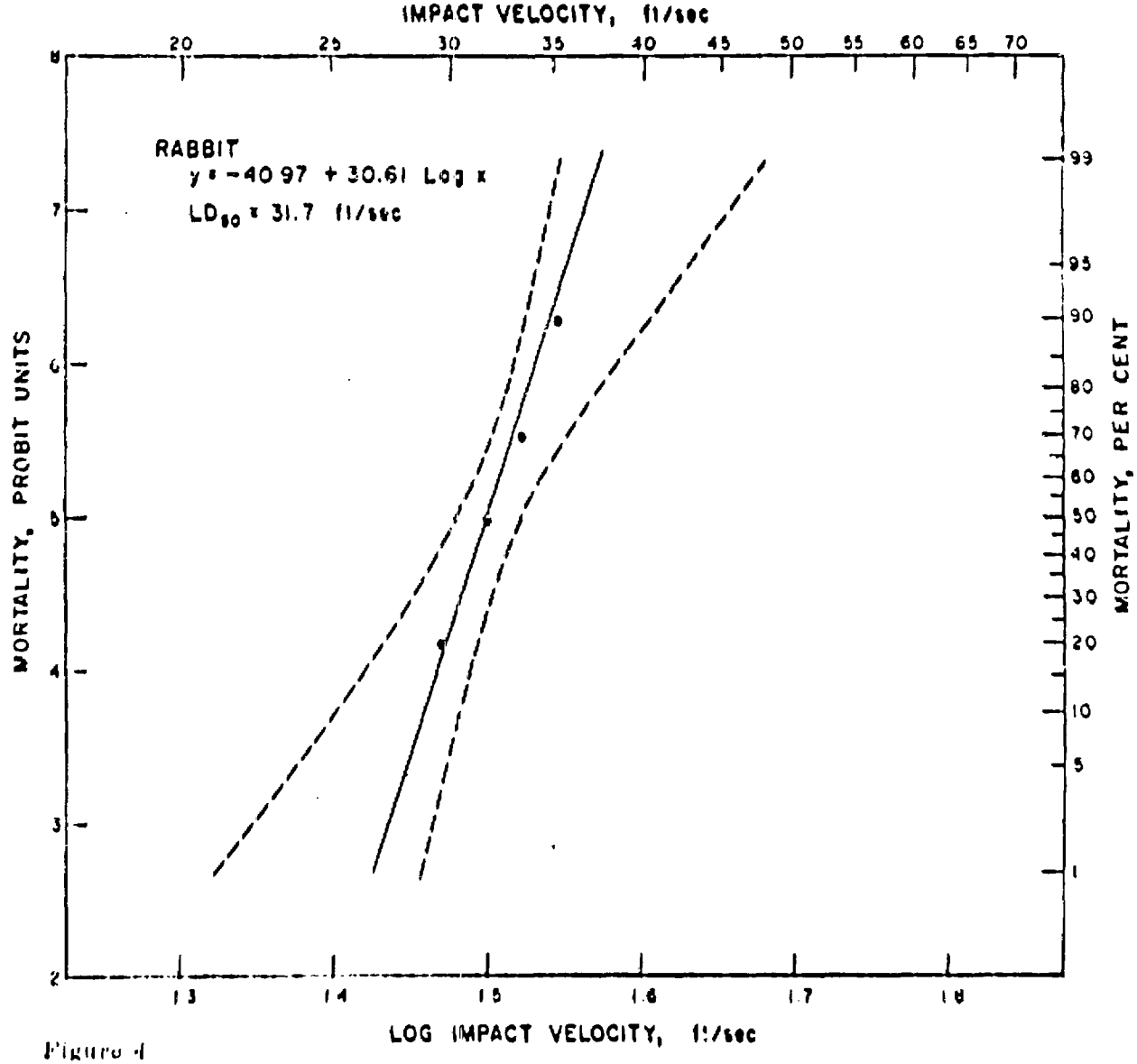
IMPACT VELOCITY, ft/sec
20
25
30
35
40
45
50
55
60
65
70
RABBIT
y = -40.97 + 30.61 Log x
LD50 = 31.7 ft/sec
MORTALITY, PROBIT UNITS
8
7
6
5
4
3
2
MORTALITY, PER CENT
99
95
90
80
70
60
50
40
30
20
10
5
1
1.3
1.4
1.5
1.6
1.7
1.8
LOG IMPACT VELOCITY, ft/sec

Figure 4

Table 6

RESULTS FROM THE PROBIT ANALYSIS

Species	Impact velocities, ft/sec, computed for*:			Probit equation constants**		
	LD_{10}	LD_{50}	LD_{90}	a (intercept)	b (slope)	S(b)***
Mouse	32.3 (27.2-34.6)	39.4 (37.4-42.0)	47.9 (44.1-59.4)	-18.86	14.96	3.02
Rat	37.4 (34.2-39.3)	43.5 (42.0-44.8)	50.7 (48.7-54.2)	-26.73	19.36	2.76
Guinea pig	27.7 (25.4-28.9)	31.0 (30.0-31.9)	34.7 (33.5-37.4)	-33.84	26.04	4.49
Rabbit	28.8 (25.0-30.3)	31.7 (30.2-33.3)	35.0 (33.3-40.1)	-40.97	30.61	7.08

*The numbers in parentheses are the 95 per cent confidence intervals.

**See Figs. 1, 2, 3, and 4 for probit equations and graphic presentations.

***The standard error of the slope constant.

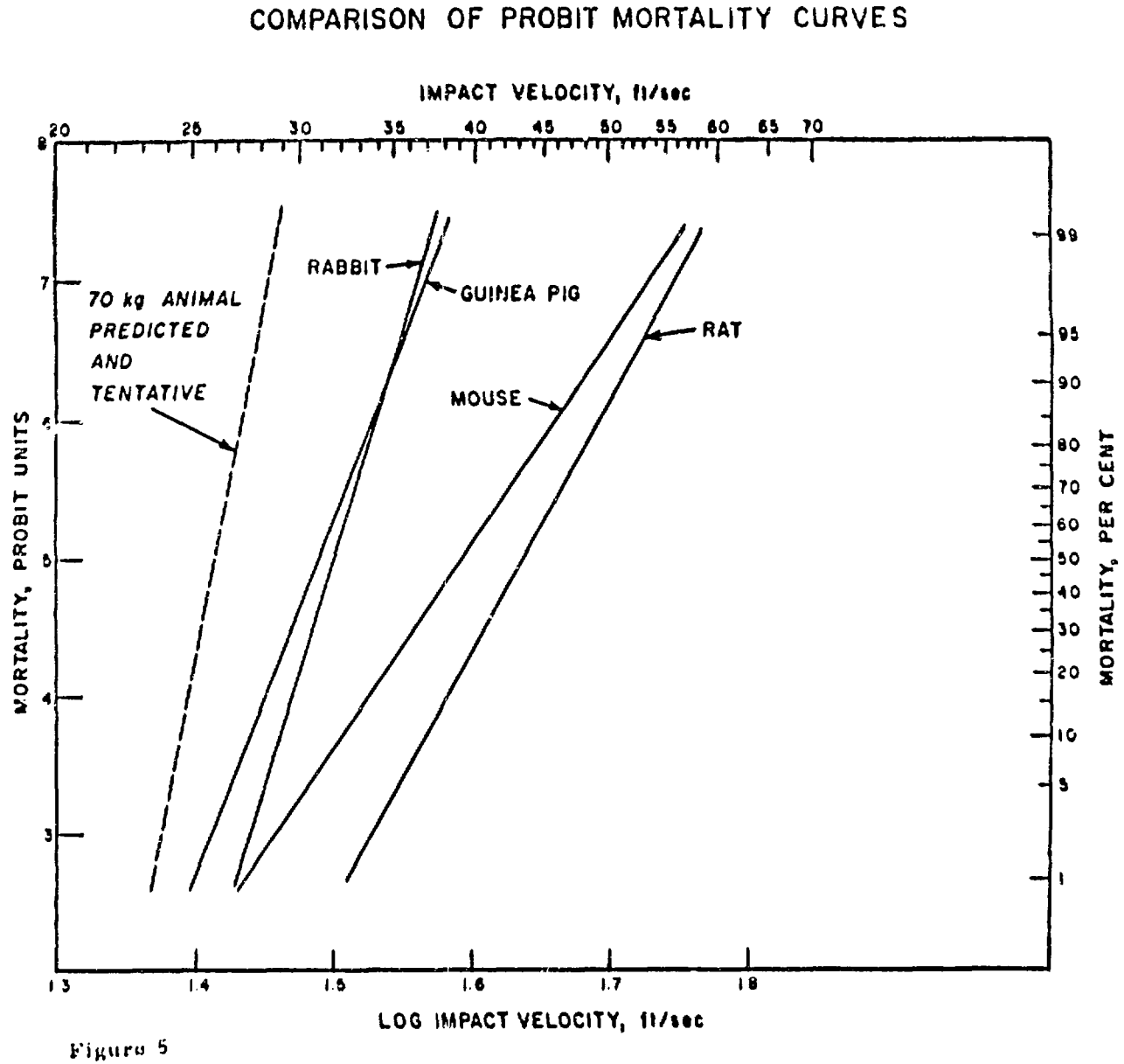
COMPARISON OF PROBIT MORTALITY CURVES
IMPACT VELOCITY, ft/sec
20
25
30
35
40
45
50
55
60
65
70
RABBIT
GUINEA PIG
RAT
MOUSE
70 kg ANIMAL
PREDICTED
AND
TENTATIVE
MORTALITY, PROBIT UNITS
7
6
5
4
3
99
95
90
80
70
60
50
40
30
20
10
5
1
MORTALITY, PER CENT
1.3
1.4
1.5
1.6
1.7
1.8
LOG IMPACT VELOCITY, ft/sec

Figure 5

that a test for parallelism using all the data indicated the results could not be fitted to a common slope with any statistical reliability. However, at the 95 per cent confidence limit, as might be expected from a visual inspection of Fig. 5, the regression curves for the mouse and rat were essentially parallel; so also were those for the guinea pig and the rabbit. Not so evident from visual inspection was the fact that the curves for the rat and guinea pig, and the rat and the rabbit could be regarded statistically as parallel. This is not the case for the mouse-rabbit and the mouse-guinea pig relationships which showed no parallelism statistically in the regression lines at the 95 per cent confidence limit.

2. Time of Death

Two hundred animals were lethally injured by impact. The number of animals succumbing in various time intervals — 0-5, 6-10, 11-20, 21-60, 61-120 minutes, and 121 minutes to 24 hours — is shown in Table 7, along with total percentage and accumulative percentage figures for the selected periods of time. Table 8 presents the percentage and accumulative percentage data for each species of animal.

The combined results given in Table 7 show that death occurred quite rapidly; e.g., 149 of the animals, or 74.5 per cent, were dead within 20 min and 179, or 89.5 per cent, within one hour. Thus, only 21 of the 200 fatally injured animals lived longer than one hour and these — about 10 per cent of the total — died within 24 hr after impact; 5 between the first and second hour and 16 between the second and twenty-fourth hour.

The species-segregated data in Table 8 show other findings of interest. First, it is apparent that the mice died within an extraordinarily short period; i.e., 52, 86, and 100 per cent were dead within 5, 10, and 20 min, respectively. Second, mortally injured rabbits survived longer than the other species. Third, the times of death for guinea pigs and rats fell between those for mice and rabbits. Fourth, at the higher accumulative percentages of lethality — above 90 per cent for all species — there was a tendency for time of death to be related to animal size; i.e., the larger the animal the longer the survival period.

Table 7

TIME OF DEATH AND

NUMBER OF ANIMALS MORTALLY WOUNDED BY IMPACT AND
THE TOTAL INCIDENCE OF MORTALITY AS A FUNCTION OF TIME

Species of animal	Number of animals dying in indicated time intervals						
	0-5 min	6-10 min	11-20 min	21-60 min	61-120 min	121 min -24 hrs	Totals
Mouse	23	15	6	0	0	0	44
Rat	22	14	12	16	2	7	73*
Guinea pig	30	6	6	9	1	5	57
Rabbit	4	4	7	5	2	4	26
Total number	79	39	31	30	5	16	200
Total per cent	39.5	19.5	15.5	15	2.5	8.0	100
Accumulative No.	79	118	149	179	184	200	
Accumulative %	39.5	59	74.5	89.5	92.0	100	

*There were 17 rats not included in the total because time of death was not recorded.

Table 8

PERCENTAGE AND ACCUMULATIVE PERCENTAGE OF LETHALLY WOUNDED ANIMALS AS A FUNCTION OF TIME AFTER IMPACT

	Percentage and Accumulative Percentage of Lethally Wounded Animals							
	Mice		Rats		Guinea pigs		Rabbits	
Time of death	%	Accum.	%	Accum.	%	Accum.	%	Accum.
0-5 min	52.3	52.3	30.1	30.1	52.6	52.6	15.4	15.4
6-10 min	34.1	86.4	19.2	49.3	10.5	63.1	15.4	30.8
11-20 min	13.6	100	16.4	65.7	10.5	73.6	26.9	57.7
21-60 min			21.9	87.6	15.8	89.4	19.2	76.9
61-120 min			2.8	90.4	1.9	91.3	7.7	84.6
121 min - 24 hrs			9.6	100	8.7	100	15.4	100

To emphasize these points Fig. 6 was prepared and shows the accumulative percent of animals mortally wounded, as given in Table 8, as a function of time of death for each species separately. Because the number of animals surviving in the longer time periods was small and because of the wide variability among species, no detailed statistical assessment of the time of death data was undertaken. However, the early time to death is quite clear and impressive.

3. Interspecies Relationships and Extrapolation of Data

a. Impact velocity and 50 per cent mortality

The interspecies relationship between the impact velocity associated with 50 per cent mortality in mice, rats, guinea pigs, and rabbits and the average weight of each species of animal was examined using the method of least squares. The results, plotted in Fig. 7, show the LD_{50} impact velocity for each species as a function of mean body weight and the regression equation which best fits the data; namely,

$$\log Y = 1.6961 - 0.0572 \log X$$

where

Y = impact velocity for 50 per cent mortality in ft/sec

X = mean body weight in grams

the intercept = 1.6961 and

the slope constant = −0.057

The standard error of the estimate was 0.042 log units (9.7%).

This regression relationship may be used tentatively to predict the impact velocity associated with 50 per cent mortality for other species of animals. Solving the equation for an animal weighing 70 kg (154 lbs) yielded a figure of 26.2 ft/sec (17.8 mph) as the predicted LD_{50} impact velocity.

b. Slopes of the mortality curves

It was of interest to explore the possible association between the average weights of the animals studied and the slopes of the probit regression

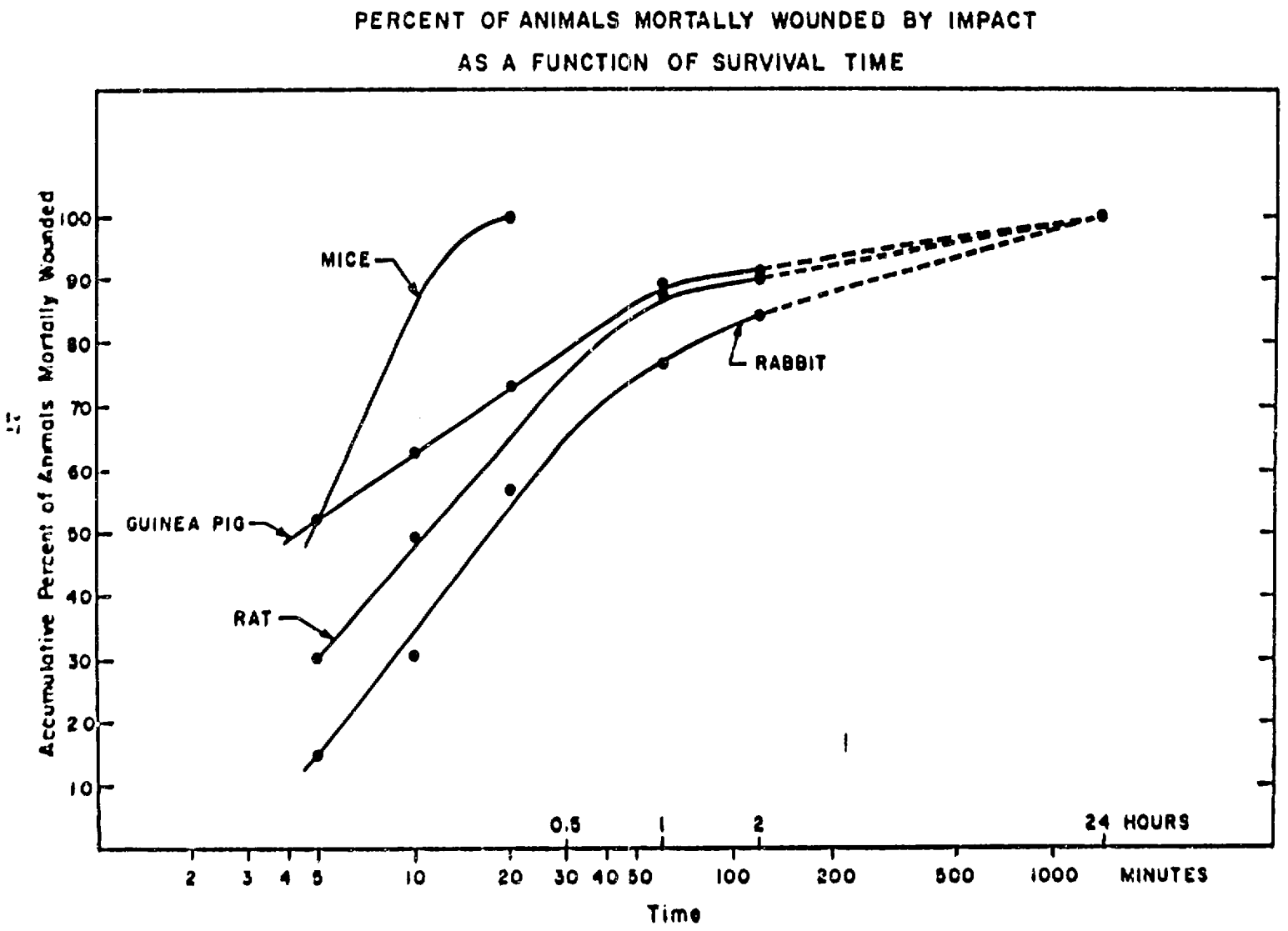
PERCENT OF ANIMALS MORTALLY WOUNDED BY IMPACT
AS A FUNCTION OF SURVIVAL TIME
Accumulative Percent of Animals Mortally Wounded
100
90
80
70
60
50
40
30
20
10
MICE
GUINEA PIG
RAT
RABBIT
0.5
1
2
24 HOURS
2
3
4
5
10
20
30
40
50
100
200
500
1000
MINUTES
Time

Figure 6

IMPACT VELOCITY ASSOCIATED WITH 50 PERCENT MORTALITY
AS A FUNCTION OF AVERAGE BODY WEIGHT

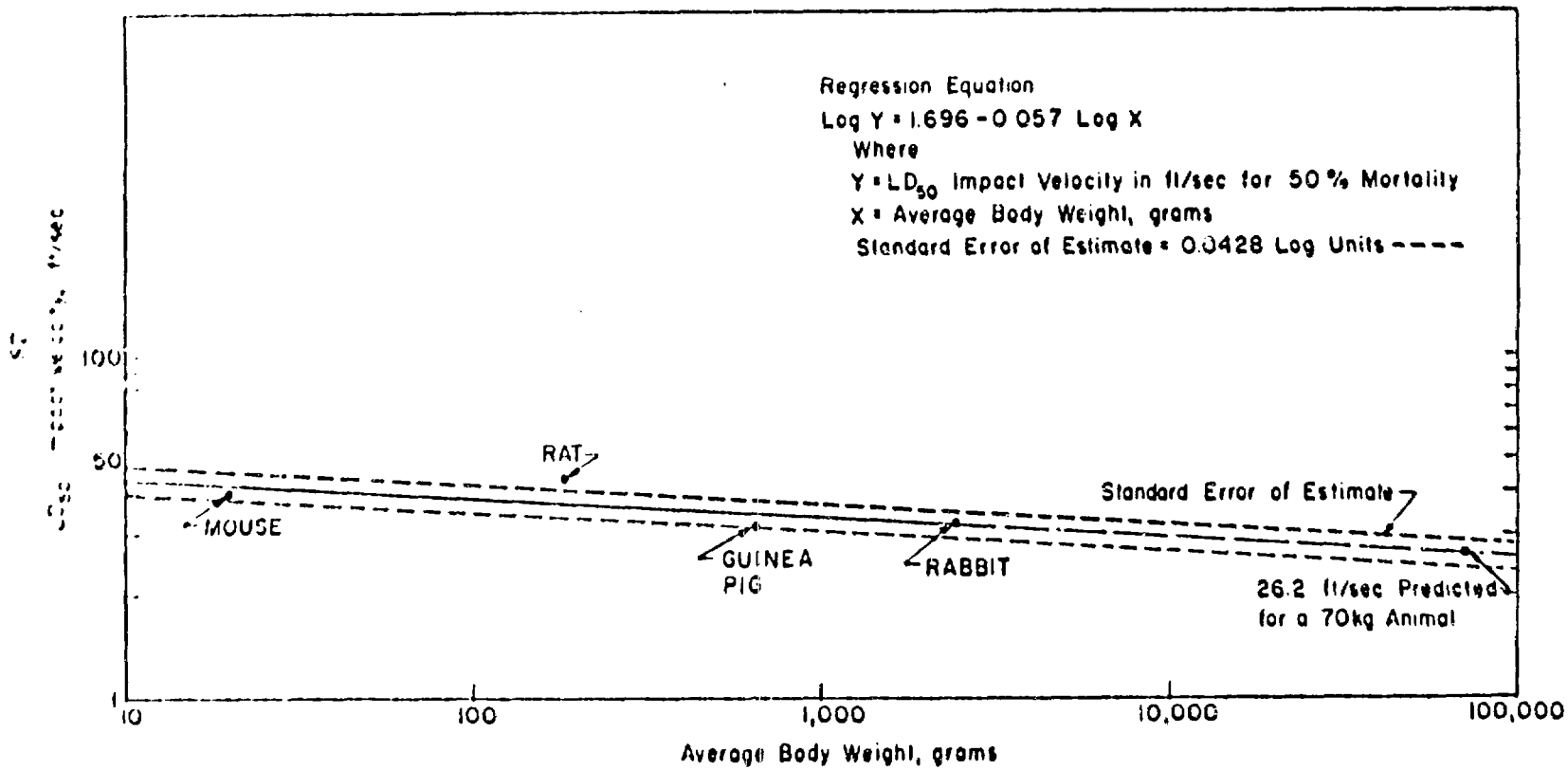

Figure 7

equations describing the empirical relationship between impact velocity and mortality. This was done using the method of least squares and a regression equation derived. The equation was:

$$\log S = 0.966 + 0.15358 \log \overline{M}$$

where

S = slope of the regression equation

$\overline{M}$ = the average body weight in grams

The standard error of the estimate was 0.017 log units (3.89%).

Solving this equation for an animal weighing 70 kg (154 lbs) yielded a predicted slope constant of 51.3. Graphic portrayal of the data relating the regression equation, slope constant, and average body weight for mice, rats, guinea pigs, and rabbits is presented in Fig. 8 along with the regression line and the extrapolation to an animal weighing 70 kg.

c. Derivation of regression equation relating impact velocity and mortality for a 70 kg animal

Having a predicted slope constant and a predicted LD_{50} impact velocity for a 70 kg animal made it a simple matter to substitute values in the regression equation of the form

$$Y = a + b \log X$$

and determine the intercept, a, of a predicted regression equation for the 70 kg animal; e.g.,

$$5 = a + 51.3 \log 26.2$$

$$a = 5 - 51.3 \log 26.2 = -67.758$$

Thus, it was possible to write for the 70 kg animal the following equation:

$$Y = -67.76 + 51.3 \log X$$

where

Y = percent mortality in probit units

X = the impact velocity in ft/sec

The regression line for the above equation is shown dotted in on Fig. 5 and

SLOPE CONSTANTS OF THE REGRESSION EQUATIONS RELATING MORTALITY AND IMPACT VELOCITY AS A FUNCTION OF ANIMAL WEIGHT

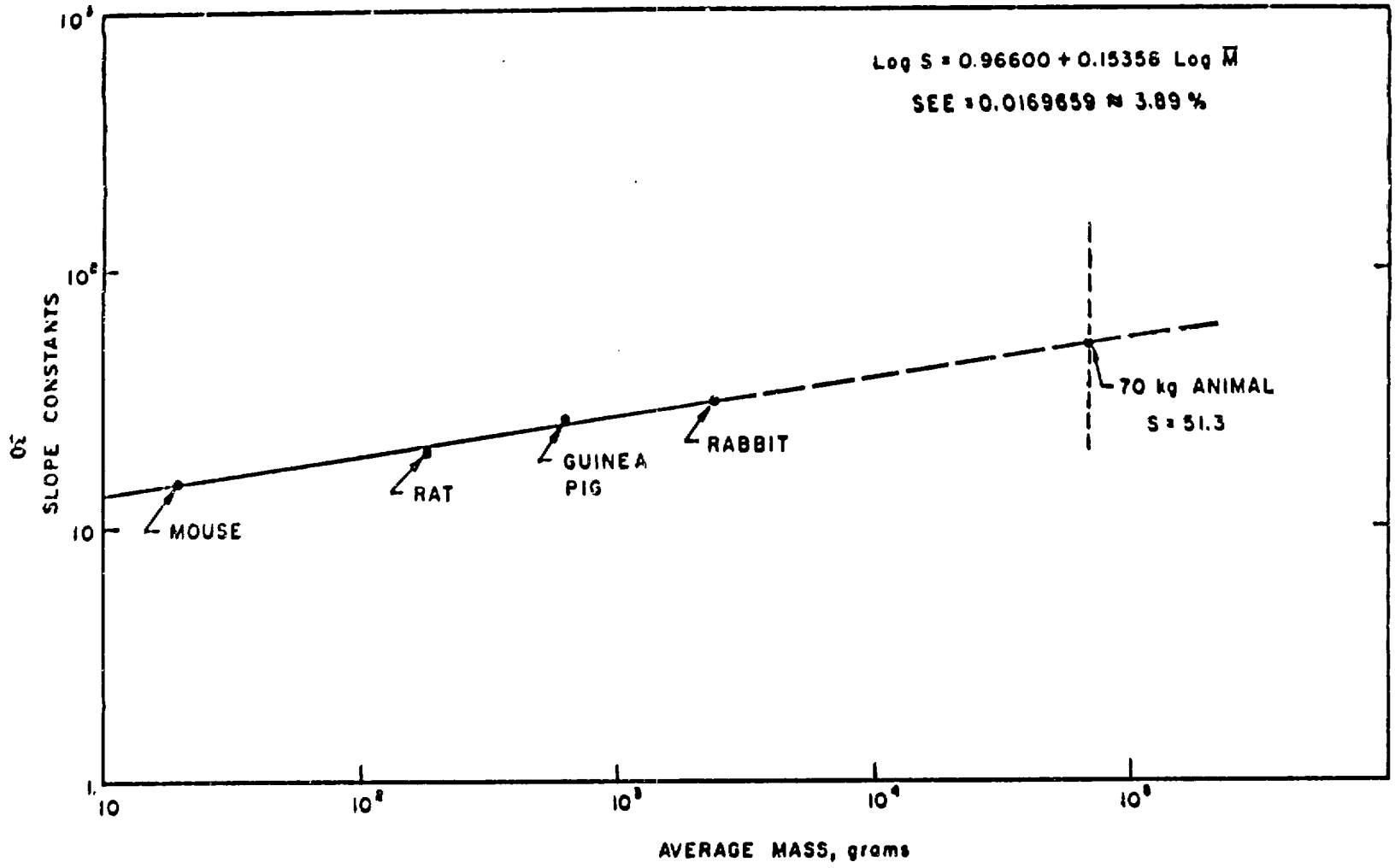

Figure 8

allows one to visualize the predicted data along with the empirical findings for mice, rats, guinea pigs, and rabbits described previously.

DISCUSSION

1. General

Strictly speaking, the data reported above apply only to young adult animals subjected to impact with a solid, flat surface in the prone position. Besides the innate biological variability mentioned years ago by Rushmer[18-20] and Rushmer et al.,[21] the experiments described here involve two other factors which might spuriously influence the relationship between mortality and impact velocity. The first concerns some variation in the position of the animals when striking the concrete surface since the righting reflexes were employed to maintain a feet-down position. The second concerns a possible modification of the impact velocity by whatever resistance the legs of the animals offered as energy absorbers to decrease the velocity of contact of the main mass of the body. Viewing the many movies taken of impact, however, revealed that in no observed instance was there much of a head- or tail-down position at impact; also, there was no appreciable slowing down of the animal detectable when velocities within the mortality range were reached.

Unfortunately, should a human be subjected to impact either involving falls, vehicular accidents, ground shock imparted to blast protective shelters or abrupt deceleration after displacement by blast winds, it is likely that considerable variation in the body area of impact will occur. Also, there are many circumstances in which a decelerative experience may involve glancing contact with an object; too, a great variation in the shape, weight and consistency of the decelerating object or surface may be involved. Any modification of the time of deceleration and the distance over which it occurs will markedly influence the magnitude of the G load and the rate with which it develops. Such factors are responsible for human survival after falls described in the well known paper of DeHaven[11] which concerned drop distances in three cases of 55, 93 and 145 ft, impact velocities ranging from about 60 to near 85 ft/sec, and stopping distance of about 0.3 to 0.7 ft occurring in a time period in the vicinity of 0.01 to 0.02 sec. Frequently, the surface struck is soft ground and

the impact area of the body is large – the back, side or ventral surface – and these factors modify the relationships between impact velocity and biological effect.

Though refinements in terms of stopping distance and time as they influence G loading are important and have been well discussed by Rushmer et al.,[20] DeHaven,[22] Roth,[23] Haddon and McFarland,[24] Stapp,[25] Goldman and von Gierke[26] and others, there is nonetheless a problem in the human case – as noted in the Introduction – when impact with a flat, solid surface occurs and the stopping times and distances are controlled only by the tissues of the body itself. Ideally, one would like to know the relationship between impact velocity and mortality, the threshold for mortality and the threshold for tolerable trauma for the human case, all as functions of the different areas of the body that may come in violent contact with hard surfaces. Fortunately, there are a few relevant data on some aspects of this problem that are helpful, first, in setting quantitative relationships for man and second, in evaluating the extrapolations set forth in the present study. The more important of these now known to the authors will now be briefly noted.

2. Literature Involving Human Material

a. Head

Black et al.,[27] reviewing the records of British mine accidents in 1942, stated a skull fracture occurred from a probable fore- and -aft blow of 15 ft/sec (equivalent to a 3-1/2 ft drop) from a striking mass of about 8 lb. Zuckerman and Black,[28] using monkeys strapped against a heavy plate set in sudden motion by the impact of a heavy pendulum, failed to produce signs of concussion or fracture with "initial" velocities of 10 ft/sec applied fore and aft.

Draeger et al.[29] ran two tests on an impact-shock test machine using cadavers lying face down and face up on the table at the time a maximum blow from a striking hammer produced an "initial" average velocity of near 15 ft/sec. It was noted that no bone damage was produced for the face-up condition in contrast to the face-down instance wherein a linear fracture of the vault of the skull in the occipital region was found.

Gurdjian et al.[30] have pointed out that dry skulls have been fractured with energies as little as 25 ft lbs (300 in. lbs), but that cadaver heads with scalp and contents intact to "cushion" the blow required energies of close to 400 to more than 900 in. lbs to fracture. Important also is the fact that 10 to 20 per cent additional energy over that required to produce a single linear fracture almost completely demolished the skull shattering it to fragments.

The same authors[31] reported experiments from which the 400 - 900 in. lbs figures were derived and pointed out the impact velocities involved when 46 intact human heads were dropped on a hard surface. These ranged from one instance with fracture at 13.5 ft/sec to about 23 ft/sec. The data grouped according to impact velocities are shown in Table 9.

While the skull varies in its strength, being minimal for mid-frontal blows and maximal for the anterior interparietal positions, and energy at impact is the more precise means of assessing tolerance to abrupt deceleration, the tabulated distribution of impact velocities required for fracture has great appeal for its simplicity. However, in assessing the data noted in Table 9, it must be realized that impact with a 90 degree sharp corner may require only 60 in. lbs of energy[32] for skull fracture and that an individual travelling horizontally and undergoing a head-on impact involves a situation different from the circumstances described above; e.g., the head then will have to absorb not only its own energy of motion, but also that of the following body as well; this also places considerable strain on the neck and cervical spine.

The careful reader will realize that nothing yet has been said about cerebral concussion. Indeed, it is true that concussion may well be a more dangerous lesion than skull fracture; too, it can occur in the absence of fracture of the cranial vault. It is unfortunate that no significant amount of quantitative human data are available for concussion,[24] though Lissner and Evans[33] have stated that if the energy to be dissipated by impact loading of the skull is kept below 400 in. lbs (33 ft lbs), they feel neither severe concussion nor fracture will result. In terms of a 10 lb mass, near the average weight of the adult human head, this is equivalent to a drop from a height of 40 in. and an

Table 9

THE RANGE OF IMPACT VELOCITIES ASSOCIATED WITH EXPERIMENTAL SKULL FRACTURE OF THE SKULLS OF INTACT HUMAN HEADS

(After Gurdjian et al.[31])

Range impact velocities ft/sec	Approx. velocity in mph	Approx. height of fall ft	Number of fractures		
			of Heads	in per cent	accumulative per cent
13.5 - 14.9	9.5	37	9	19	19
15 - 16.9	10.9	48	10	22	41
17 - 18.9	12.2	61	12	26	67
19 - 20.9	13.6	75	13	24	91
21 - 22.9	15.0	91	4	9	100
Totals			46	100	

Minimum velocity with fracture - 13.5 ft/sec (9.2 mph)

Maximum velocity with fracture - 22.8 ft/sec (15.5 mph)

Maximum and minimum velocity without fracture - unstated

impact velocity of 14.7 ft/sec. This figure is well above the British experience of Zuckerman and Black[28] with monkeys, quoted above, noting that 10 ft/sec produced no signs of concussion or fracture.

Last, with regard to the head problem, no data are at hand for infants, children and adolescents at one end of the age scale nor those in the last decades of life at the other as pointed out by Haddon and McFarland[24] in a competent general review of the present knowledge concerning head injury. However, for adults the consistency between the British and American data placing the threshold for skull fracture at near 13 ft/sec allows one to feel fairly confident that an impact velocity with a hard, flat surface of 10 ft/sec should prove to be an acceptable impact velocity for the head of adult man which opinion is compatible with findings attributed to Lombard;[23] namely, that helmeted subjects voluntarily tolerated blows to the helmet, involving velocities from about 11 - 14 ft/sec. Such blows involved an acceleration distance of near 0.1 ft, force application time close to 17 msec and a maximum G load of from 15 to 35 G.

b. Lower extremity

Casualty experience during the second World War included many instances of the very serious fracture of the calcaneus (heel bone), other bones of the foot, legs, spine, and skull which were caused by explosions of bombs, mines, or torpedoes below the decks of or near vessels.[34, 35] Such observations stimulated laboratory investigations on the lower extremity of intact cadavers.

In Great Britain, Black, Christopherson, and Zuckerman[27] reported experiments in 1942 using two embalmed cadavers. With the knees locked and with the bottoms of the feet made parallel with the floor, using wooden blocks, one of the cadavers was dropped to the deck from heights of 0.5, 1.0, 2.0, and 4.0 ft. Only the latter drop produced boney pathology — a complete fracture of the heel bones bilaterally with a "chip fracture" in the posterior surface of each. The impact velocities at 2 and 4 ft were about 11 and 16 ft/sec, respectively, and the authors concluded that an initial velocity within these limits might well mark the fracture threshold for barefooted individuals.

The second cadaver was fitted with "specially devised boots which had sponge-rubber pads on the inside of strong rubber heels". After a drop of 3 ft (near 13.9 ft/sec impact velocity) fracture of the left talus bone was noted (the talus lies above the calcaneus, or heel bone, and separates the latter from the two bones of the lower leg at the ankle). After drops from 6 ft (19.6 ft/sec) additional fractures were noted; e.g., inner margins of the lower end of the left tibia; the outer and inner condyle of the upper end of the left tibia, and the whole upper end of the right tibia.

In 1945 Draeger et al.[29] described experiments with four embalmed cadavers and human volunteers using a high impact test machine, the 4,000 lb table of which was energized by an upward blow of a 3,000 lb hammer allowed to swing in an arc from different heights. Fractures were produced in two of the cadavers under circumstances for one covered by high speed photography. The impact velocities withstood by human volunteers was not stated.

The photographic records revealed the data noted in Table 10 showing the movement of the table on which the cadaver was standing with knees locked and the average velocities of the table and a metal bar piercing the tibia just above the ankle of the subject, both given as a function of time. Fractures of the os calcis (calcaneus) occurred and the reader will note that over the first 5 msec the velocity figures given in the next to last column of Table 10, obtained by step-by-step calculations for table movement, ranged from 12.9 to 21.4 ft/sec. These numbers are reasonably close to the British figures which placed the "initial velocity" threshold for fracture of the heel bone between 11 and 16 ft/sec.

Though there is much food for thought in the work of Draeger et al., in the interest of simplicity it is well to emphasize that impact velocities much above 11 - 12 ft/sec can cause fracture. In relation to these data for fractures, it is appropriate now to direct attention to recent work with human volunteers which goes to the point of voluntary tolerance to vertical loads applied to the feet of standing human volunteers.

Swearingen et al.[36] have reported nearly 500 experiments with 13

Table 10

IMPACT TABLE MOVEMENT AT DIFFERENT TIMES AND THE AVERAGE VELOCITIES OF THE TABLE TOP AND THE TIBIA OF A CADAVER EXPOSED STANDING WITH KNEES LOCKED

(After Draeger et al.[29])

Displacement of table in.	Time in msec	Average velocity in ft/sec	
		Table top	Tibia
0 -0.25	0 -1.62	12.9	9.8
0.25-0.60	1.62-3.25	16.9	10.1
0.60-1.50	3.25-5.0	21.4	16.2
1.05-1.09	5.0 -6.5	2.2	-5.0
1.09-1.33	6.5 -8.2	11.8	9.8
1.33-1.73	8.2 -10.0	18.5	18.1
0 -2.48	0 -15	13.7	10.2 smoothed curve

adults subjected to drop tests in a track-guided chair travelling vertically downward to impact against a platform. The movement of the latter was damped with heavy leaf springs and hydraulic pistons. Though the base platform was capable of a maximal movement of 1 in., the actual movement at impact was known to be small, but not stated. However, G-time recordings were made when standing individuals with knees locked were subjected to drops from a maximal height of 2 ft* The theoretical impact velocity connected with this fall height is 11.3 ft/sec. Integration of the G-time curve recorded and reported — which showed a maximum G of 65 developing at 10,000 G/sec with impact enduring for 8 msec — gave a calculated impact velocity of 9.9 ft/sec. This figure is within about 12 per cent of the theoretical figure.

The loading associated with about 10 ft/sec impact velocity was the maximal tolerated by the human subjects. Severe pain was noted in the chest, epigastrum, lower back, hip joints, and top of the head. Also, pain was reported in the arches of the feet, back of the legs, ankles, heels, and throat.

c. Spine

In similar experiments with seated subjects, Swearingen and coworkers[36] determined the limit of voluntary tolerance to be associated with a maximal load of 95 G developing at 19,000 G/sec over a time period of 7.5 msec; the impact velocity calculated from the G-time curves was 9.7 ft/sec. Severe pain in the chest, spine, head, and stomach was noted and "Shock: severe, general" was reported.

There is little point in reviewing the many ejection seat data considered safe and unsafe by various investigators. Let it suffice to say that they are not inconsistent with the findings of Swearingen et al., that Ruff[37] estimated fractures of the spine could occur at about 100 G when the time involved was as short as 2 msec, and that Gagge and Shaw[38] have stated application of 20 G developing at the rate of 150 G/sec and enduring for 200 msec was acceptable for pilots using ejection seats for escape from aircraft.

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*Swearingen, J. J., personal communication.

and that Watts et al.[39] reported 20 G for 0.08 sec applied at the rate of 200 G/sec produced no symptoms in 50 volunteer naval subjects.

d. Automobile accidents

Finally, it is of considerable interest to note National Safety Council statistics quoted by DeHaven[22] relevant to fatalities in urban automobile accidents. The figures show that "40 per cent of automobile fatalities in urban areas involved a speed of 20 mph or less and 70 per cent were attributed to accidents in which the speed did not exceed 30 mph." This would place the 50 per cent mortality figure near 23 mph (33.8 ft/sec). It is necessary to point out, however, that this velocity apparently refers to the speed at which a crash occurred and may or may not refer to actual velocity at which a fatally injured person struck a solid surface.

3. Present Study

a. General

Obviously what has been assembled from the literature both for the human and the animal case, along with the present interspecies study, indicates that the "state of the art" for understanding the biology of decelerative impact is not very far advanced. Much more quantitative information is needed to establish tolerance for various organs and regions of the body, particularly in the case of the friable liver and spleen and the other abdominal organs. Likewise, additional data are desired for the thorax and its organs, for the head and its contents, and for the cervical spine. Be this as it may, a few comments are in order concerning the experiments reported here and their relation to the literature reviewed. These will now be presented.

b. Extrapoloation of the LD_{50} impact velocity data

Though it is hardly possible to imagine what precise use might be made of the described interspecies extrapolation of the LD_{50} impact velocity to give a figure of 26 ft/sec (18 mph) for the 70 kg animal, it is none the less quite interesting that the data for human fatalities in automobile statistics[22] show a 50 per cent mortality at vehicular speeds near 33.8 ft/sec (23 mph). Thus, the animal extrapolation of the 50 per cent impact velocity is 22.5 per

cent lower than the vehicular speeds associated with 50 per cent fatalities. While this apparent correspondence may be more fortuitous than real and a number of grave uncertainties are no doubt involved, it could also represent more than an accidental array of factors. At least, the situation is sufficiently encouraging to suggest a number of worth while contingencies. First, additional and somewhat similar animal studies are justified; second, all efforts to collect relevant data referable to the human case from past experience and in the future are indicated; third, the extrapolation to the 70 kg animal can be tentatively regarded as applying "on the average" to man (a) for the purposes of testing such a hypothesis, and (b) for use under certain circumstances because nothing better seems to be at hand.

c. The regression equation for the 70 kg animal and the threshold for mortality and injury concept

The regression equation for the 70 kg animal — arrived at by extrapolation and predicting the relationship between impact velocity and mortality — is of interest, for with its use one can explore the mortality threshold situation for the 70 kg animal as well as for the four species empirically studied. Assigning zero to Y in the probit regression equations and solving them for X, yields figures for impact velocities predicted to be near the threshold for mortality. Doing this simple calculation gave the figures set forth in Table 11.

Two things are significant about the tabulated data in Table 11. First, there is very little difference in the threshold impact velocities for all species and for the 70 kg animal, suggesting there may be a common mechanism that is critical for mortality. Second, the impact-velocity numbers are higher than those known to be associated with quite dangerous, perhaps fatal, lesions in man, such as the range in impact velocities for human skull fracture from 13.5 to 23 ft/sec (9.2 - 15.6 mph) reported by Gurdjian et al.[31] Third, the predicted impact velocities for the threshold of mortality are well above the impact velocity of about 10 ft/sec voluntarily tolerated by standing and seated human subjects studied by Swearingen.[36] Fourth, the general consistency of the information just noted above suggests one can tentatively take

Table 11

PREDICTED IMPACT VELOCITY AT THRESHOLD OF MORTALITY

Animal Species	Predicted impact velocity at mortality threshold	
	ft/sec	mph
Mouse	18.2	12.8
Rat	24.0	16.3
Guinea pig	19.9	13.5
Rabbit	21.8	14.8
70 kg animal	20.8	14.1

10 ft/sec as "an-on-the-average safe" impact velocity for adult humans and regard the probabilities of serious injury and even fatality for man to increase progressively as the impact velocity is elevated above this figure.

d. Time of death

It is well to reemphasize again the short time to death observed in the 200 untreated animals dying of impact in relation to the high mortality figures associated with vehicular accidents which reoccur on an annual basis. How many of the animals dying in the present study could have been saved by therapeutic measures is, of course, not known, but there are many human accident victims alive today because medical care was appropriate both in kind and in time. The rapidity with which the experimental animals expired makes it impossible to resist suggesting that one possible way to reduce fatalities in vehicular accidents would be to explore and implement all arrangements that would assure the earliest possible medical care.

e. Cause of death

Finally, the inquisitive reader can well ponder along with the authors the several possible pathophysiological mechanisms responsible for death of the animals studied. Currently, it is not possible to present relevant data; neither may it be possible to do so in the future. However, gross pathological observations were made on the animals who died spontaneously and who were sacrificed after impact. It remains for further studies to reveal whether the gross data are adequate or inadequate to the challenge of throwing more light on the etiology of death by violent impact.

REFERENCES

1. White, C. S.; Chiffelle, T. L.; Richmond, D. R.; Lockyear, W. H.; Bowen, I. G.; Goldizen, V. C.; Merideth, H. W.; Kilgore, D. E.; Longwell, B. B.; Parker, J. T.; Sherping, F.; and Cribb, M. E., "The Biological Effects of Pressure Phenomena Occurring Inside Protective Shelters Following Nuclear Detonation," Operation Teapot Report, WT-1179, October 28, 1957.

2. White, C. S. and Richmond, D. R., "Blast Biology," Technical Progress Report, TID-5764, Lovelace Foundation for Medical Education and Research, Albuquerque, New Mexico, September 18, 1959.

3. White, C. S. and Richmond, D. R., "Blast Biology," Chapter 63 in Clinical Cardiopulmonary Physiology, Grune and Stratton, New York and London, 1960.

4. White, C. S.; Bowen, I. G.; Richmond, D. R.; and Corsbie, R. L., "Comparative Nuclear Effects of Biomedical Interest," CEX-58.8, Civil Effects Test Operations, U. S. Atomic Energy Commission, January 12, 1961.

5. White, C. S., "Biological Blast Effects," TID-5564, Lovelace Foundation for Medical Education and Research, Albuquerque, New Mexico, September, 1959.

6. Desaga, H., "Blast Injuries," Chapter XIV-D, pp. 1274-1293, in German Aviation Medicine, World War II, Vol. II, U. S. Government Printing Office, Washington, 1950.

7. Benzinger, T., "Physiological Effects of Blast in Air and Water," Chapter XIV-B, pp. 1225-1259, in German Aviation Medicine, World War II, Vol. II, U. S. Government Printing Office, Washington, 1950.

8. Clemedson, Carl-Johan and Hultman, H., "Air Embolism and the Cause of Death in Blast Injury," Military Surgeon 114:424-437, 1954.

9. Ross, Joan M., "Haemorrhage in the Lungs in Cases of Death Due to Trauma," Brit. Med. J. 1:79, January, 1941.

10. Osborn, G. R., "Findings in 262 Fatal Accidents," Lancet 2:277, September, 1943.

11. DeHaven, Hugh, "Mechanical Analysis of Survival in Falls from Heights of Fifty to One Hundred and Fifty Feet," War Med. 2:586-596, July, 1942.

12. Teare, Donald, "Postmortem Examinations on Air-Crash Victims," Brit. Med. J. 2:707-708, September, 1951.

13. Hass, G. M., "Types of Internal Injuries of Personnel Involved in Aircraft Accidents," J. Aviat. Med. 15:77-84, April, 1944.

14. Taborelli, R. V.; Bowen, I. G.; and Fletcher, E. R., "Tertiary Effect of Blast — Displacement," Operation Plumbbob Report WT-1469, May 22, 1959.

15. Bowen, I. G.; Albright, R. W.; Fletcher, E. R.; and White, C. S., "A Model Designed to Predict the Motion of Objects Translated by Classical Blast Waves," Civil Effects Test Operations, CEX-58.9 (in press).

16. Fletcher, E. R.; Albright, R. W.; Goldizen, V. C.; and Bowen, I. G., "Determinations of Aerodynamic Drag Parameters of Small Irregular Objects by Means of Drop Tests," Progress Report, submitted to Division of Biology and Medicine, U. S. Atomic Energy Commission, October 11, 1960.

17. Finney, D. J., Probit Analysis. A Statistical Treatment of the Sigmoid Response Curve, 2nd Edition, Cambridge University Press, 1952.

18. Rushmer, R. F., "Internal Injury Produced by Abrupt Deceleration of Small Animals," Report No. 1, Project 241, AAF School of Aviation Medicine, Randolph Field, Texas, September 2, 1944.

19. Rushmer, R. F., "Comparison of Experimental Injuries Resulting from Decelerative Forces Applied to the Ventral and Dorsal Aspects of Rabbits During Simulated Aircraft Accidents," Report No. 1, Project 301, AAF School of Aviation Medicine, Randolph Field, Texas, October 8, 1944.

20. Rushmer, R. F., "The Changes in Pressure in the Peritoneal Cavity Produced by Sudden Deceleration of Experimental Animals," Report No. 1, Project 472, AAF School of Aviation Medicine, Randolph Field, Texas, April 19, 1946.

21. Rushmer, R. F.; Green, E. L.; and Kingsley, H. D., "Internal Injuries Produced by Abrupt Deceleration of Experimental Animals," J. Aviat. Med. 17:511-525, 1946.

22. DeHaven, Hugh, "Mechanics of Injury Under Force Conditions," Mech. Engineering 66:264-268, 1944.

23. Roth, H. P., "Impact and Dynamic Response of the Body," Symposium on Frontiers of Man-Controlled Flight (edited by H. Haber), The Institute of Transportation and Traffic Engineering, University of California, Los Angeles, April 3, 1953.

24. Haddon, W. A. and McFarland, R. A., "A Survey of Present Knowledge of the Physical Thresholds of Human Head Injury from the Engineering Standpoint," Harvard School of Public Health Report sponsored by the Commission on Accidental Trauma of the Armed Forces Epidemiological Board, Department of Defense, Boston, Massachusetts (undated).

25. Stapp, J. P., "Tolerance to Abrupt Deceleration," pp. 122-169 in Collected Papers on Aviation Medicine, AGARDograph No. 6, Butterworths Scientific Publications, London, 1955.

26. Goldman, D. E. and von Gierke, H. E., "The Effects of Shock and Vibration on Man," Lecture and Review Series No. 60-3, Naval Medical Research Institute, Bethesda, Maryland, January 8, 1960.

27. Black, A. N.; Christopherson, D. G.; and Zuckerman, S., "Fractures of the Head and Feet," Ministry of Home Security, Report RC-334, Oxford, England, August 12, 1942.

28. Zuckerman, S. and Black, A. N., "The Effect of Impact on the Head and Back of Monkeys," Ministry of Home Security Report RC-124, Oxford, England, August 12, 1940.

29. Draeger, R. H.; Barr, J. S.; Dunbar, J. Y.; Sager, W. W.; and Shelesnyak, M. C., "A Study of Personnel Injury by 'Solid Blast' and the Design and Evaluation of Protective Devices," Report No. I, Research Project X-517, U. S. Naval Medical Research Institute and U. S. Naval Hospital, Bethesda, Maryland, March 30, 1945.

30. Gurdjian, E. S.; Webster, J. E.; and Lissner, H. L., "Biomechanics: Fractures, Skull," pp. 99-105, in Medical Physics, Vol. II (Editor, Otto Glasser), The Year Book Publishers, Chicago, 1950.

31. Gurdjian, E. S.; Webster, J. E.; and Lissner, H. L., "Studies on Skull Fracture with Particular Reference to Engineering Factors," Amer. J. Surg. 78:736-742, 1949.

32. Dye, E. R., "Kinematics of the Human Body Under Crash Conditions," Clinical Orthopedics 8:305-309, 1956.

33. Lissner, H. L. and Evans, F. G., "Engineering Aspects of Fractures," Clinical Orthopedics 8:310-322, 1958.

34. Barr, J. S.; Draeger, R. H.; and Sager, W. W., "Solid Blast Personnel Injury: A Clinical Study," Military Surgeon 98:1-13, 1946.

35. Draeger, R. H.; Barr, J. S.; and Sager, W. W., "Blast Injury," J. A. M. A. 132:762-767, 1946.

36. Swearingen, J. J.; McFadden, E. B.; Garner, J. D.; and Blethrow, J. G., "Human Tolerance to Vertical Impact," Aerospace Med. 31: 989-998, 1960.

37. Ruff, S., "Brief Acceleration: Less Than One Second," Chapter VI-C, pp. 584-597, in German Aviation Medicine, World War II, Vol. I, U. S. Government Printing Office, Washington, 1950.

38. Gagge, A. P. and Shaw, R. S., "Aviation Medicine," pp. 41-65, in Medical Physics, Vol. II, The Year Book Publishers, Inc., Chicago, 1950.

39. Watts, D. T.; Mendelson, E. S.; and Kornfield, A. T., "Human Tolerance to Acceleration Applied from Seat to Head During Ejection Seat Tests," Navy Department, BuAer Report No. 1, TED No. NAM 256005, January, 1947.

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